How Stimulants Induce Nightmares and Sleep Paralysis Events Through Dopamine Oversaturation

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Introduction

Although it is common knowledge that consuming sugars or stimulants before sleep makes nightmares more likely, little has been done to determine why this is the case.

Abstract

As the brain enters a sleep state, the usage of signalling molecules called dopamines by most parts of the brain is reduced or suspended, but the production of dopamines continues at the normal rate throughout brain tissues. Dopamines are, thus, at full saturation in the brain during the state of rest.

Stimulants drive the production and use of dopamines by those parts of the brain which remain active, even during sleep. The Pre-Motor Cortex generates pseudo-visual information which is interpreted as visual information coming from the occipital lobe despite the dormancy of the occipital lobe. As described in previous publications (ibid.,) this is the basis of the visual information perceived during dreams. The purpose of this function of the Pre-Motor Cortex is to create a sort of diversion which distracts a person from actual sensory inputs which might disrupt sleep. The ability to dream; perhaps not uniquely human but certainly unique to a certain subset of animals; allows for more complete elimination of glutamate waste (neurotransmitter byproducts) which dampen finer electrical activity associated with higher reasoning. This is the reason why dreaming is so essential to quality sleep and to the higher neurological functions of which humans are capable.

Nightmares and sleep paralysis disrupt sleep in ways which are not desired. When stimulants are consumed, dopamine levels increase excessively, causing an inability in the frontal lobe to perceive when one image being presented by the PMC changes to the next. This could be metaphorically understood as a transient version of the phenomenon of “screen burn-in” associated with plasma displays.

Although the PMC continues to present variant pseudo-visual information to the frontal lobe, the oversaturation of dopamine causes receptors to be occupied, preventing the transfer of voltage potentials. This results in a dreamer having the perception that the same visual information is continuing to be presented even though new information is flowing to the frontal lobe which cannot be interpreted. To the frontal lobe, this is interpreted as paralysis, which is causes the brain, as a whole, to signal strongly that something is wrong. This results in a cascade which ultimately results, usually, in a person waking from their slumber in a sweat with their heart

racing. In the case that further dreaming occurs, it usually leads to further cycles of sleep paralysis which eventually abate when dopamine levels decrease.

Conclusion

This facet of human neurology is fascinating despite having no clear practical importance.